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NEW BEDFORD HARBOR
FEASIBILITY STUDY
TASK 22
ENVIRONMENTAL EVALUATION
ACTIVITY 22.4
SELECTION OF ADDITIONAL
CONTAMINANTS FOR INCLUSION
IN THE RISK ASSESSMENT
AND FEASIBILITY STUDY

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1.0 INTRODUCTION

This report describes the methodology used to assess the organic contamination detected in the New Bedford Harbor study area and determine if contaminants other than PCBs should be addressed in the human health and environmental risk assessment. The risk assessment and feasibility study efforts to date have focused only on PCBs and metals. However, additional contaminants have been identified within the New Bedford Harbor area.

Using the Hazardous Substance List (HSL) organic data compiled on New Bedford, Jordan defined the extent of organic contamination (location and frequency of detection) and determined, based on predicted human activities and environmental conditions, the potential for exposure to these compounds. This information was then evaluated using toxicity data to determine the potential risks associated with organic contamination within New Bedford Harbor and the need to include these compounds in the risk assessment.

Most of the organic compounds detected in sediments are collocated with PCBs and occur at concentrations less than or equal to the observed PCB concentrations. The relative toxicity of these compounds with respect to the toxicity of PCBs indicates that the risk from contaminant exposure will be attributable to the PCB contamination. However, some contaminants were observed at levels greater than the corresponding PCB concentrations and at levels which may present a public health and/or environmental concern. Identifying these contaminants and locations of high contamination was the focus of this review.

Section 2.0 is a description of the review and screening process undertaken by Jordan to determine if contaminants other than PCBs should be included in the risk assessment. Section 3.0 contains a discussion of Jordan's evaluation and findings. Section 4.0 briefly summarizes the conclusions.

2.0 APPROACH

2.1 DATA SOURCES

The majority of the organic compound data generated within the New Bedford Harbor area is from sediment samples; thus, the focus of the data review was on this medium. No Hazardous Substance List (HSL) organic biota, air, or water data were available to Jordan for this review. The New Bedford Harbor data base is incomplete, and could not be used to identify organic sampling efforts within the New Bedford Harbor area. Therefore, Jordan's review focused on the validated organics data available as of January 1987 as outlined in the New Bedford Harbor Data Status Table. Jordan reviewed the organic compound data collected by: Battelle (Hot Spot sediment sampling program); U.S. Army Corps of Engineers (Wetlands eastern shore grid sampling and Hot Spot sampling) and NUS/GZA (harbor grid sampling). These represent the most recent sampling of the estuary (including the Hot Spots) and harbor and were considered to represent present-day site conditions. A total of 151 sediment data points were reviewed for both PCBs and HSL organic compounds.

2.2 METHODOLOGY

Jordan identified the organic compounds detected at each sampling point. This review indicated that, next to PCBs, polycyclic aromatic hydrocarbons (PAHs) and phthalate ester compounds appear in both the greatest concentration and most widespread distribution within the estuary and harbor area. Although, Volatile Organic Compounds (VOC) were detected, they appeared at much lower concentrations than either PAH or phthalate ester compounds. In addition, VOCs are not considered to be persistent in the environment, or to bioconcentrate in the food chain. Thus, VOCs were not considered to contribute significantly to the overall risks, and were not evaluated further in this review. Total PCB, PAH, and phthalate ester concentrations at each sampling location are presented in Table 1.

2.2.1 Phase I Screening

An initial review of the samples contained in Table 1 was performed to identify areas where PCBs may not be the major contaminant. Sample locations where the total PAH and/or total phthalate ester concentration exceeded the corresponding PCB concentration were considered important as they could potentially remain contaminated after remediation if remedial alternatives are based solely on the extent and level of PCB contamination. Sample locations where the total PAH and/or total phthalate ester concentration exceeded the corresponding PCB concentration were considered for further evaluation. The

TABLE 1: NEW BEDFORD HARBOR SEDIMENT ORGANIC DATA
1984 to 1986

		TOTAL PCBs	TOTAL PAHs	TOTAL PHTHALATES
* SAMPLE NUMBER		(mg/kg)	(mg/kg)	(mg/kg)
AE	822	ND	ND	2.97
AD	919	ND	ND	0.25
AD	928	ND	ND	0.11
AD	838	ND	ND	0.10
AE	868	ND	ND	1.80
AD	591	ND	ND	ND
AF	818	ND	ND	8.15
AD	587	ND	ND	ND
AC	320	ND	ND	0.76
AE	824	ND	ND	0.22
AD	839	ND	ND	0.04
AF	819	ND	ND	ND
AC	345	ND	ND	15.84
AC	315	ND	ND	1.10
AC	301	ND	ND	0.47
AF	233	ND	ND	ND
AC	311	ND	ND	1.50
AF	817	ND	ND	9.27
AC	310	ND	ND	1.10
AD	597	ND	ND	ND
AD	923	ND	ND	ND
AD	572	ND	0.04	0.04
AE	821	ND	0.10	2.92
AE	870	ND	0.12	5.38
AF	231	ND	0.12	0.64
AF	542	ND	0.13	1.29
AD	560	ND	0.20	0.19
AD	561	ND	0.20	0.18
AD	564	ND	0.20	0.15
AF	230	ND	0.43	0.45
AD	588	ND	0.69	ND
AD	556	ND	0.80	27.65
AD	558	ND	1.10	18.53
AD	573	ND	1.60	0.08
AC	330	ND	1.91	1.80
AC	333	ND	2.74	1.60
AE	867	ND	5.82	1.05
AF	180	(R)	ND	1.91
AF	187	(R)	ND	ND
AF	181	(R)	ND	2.17
AF	184	(R)	0.15	1.22
AF	186	(R)	0.32	0.76
AF	182	(R)	0.48	2.60
AF	183	(R)	0.92	3.70

TABLE 1: NEW BEDFORD HARBOR SEDIMENT ORGANIC DATA
1984 to 1986
(Continued)

		TOTAL PCBs	TOTAL PAHs	TOTAL PHTHALATES
* SAMPLE NUMBER		(mg/kg)	(mg/kg)	(mg/kg)
AF	185	(R)	1.03	0.53
AD	569	0.04	0.34	0.81
AD	920	0.21	ND	ND
AE	823	0.24	0.52	3.20
AD	596	0.24	1.62	ND
AD	557	0.39	0.14	12.25
AD	925	0.42	0.05	ND
AF	541	0.52	5.93	10.87
AD	600	0.53	0.10	ND
AF	810	0.59	0.94	10.21
AF	814	0.59	9.16	10.76
AF	538	0.64	1.47	2.40
AF	229	0.66	2.11	3.05
AD	589	0.69	ND	ND
AF	801	0.74	4.01	6.37
AF	816	0.80	4.72	57.47
AE	871	0.83	0.15	3.94
AD	577	0.84	ND	21.90
AF	807	1.00	1.43	ND
AD	829	1.10	ND	ND
AD	566	1.10	ND	3.20
AF	176	1.10	21.26	92.00
AD	568	1.30	ND	0.76
AD	935	1.32	ND	1.33
AD	939	1.48	ND	ND
AD	828	1.48	7.00	ND
AD	598	1.50	11.10	ND
AE	820	1.88	0.70	2.70
AF	809	2.30	1.36	6.19
AD	929	2.40	0.63	0.79
AF	813	2.40	7.19	8.84
AD	936	2.90	0.17	0.50
AF	808	2.90	0.33	2.99
AD	581	3.10	10.58	9.39
AF	802	3.20	6.68	1.05
AF	227	3.49	17.95	5.60
AD	940	3.74	ND	ND
AF	539	3.86	1.22	4.57
AE	869	4.20	2.69	8.80
AF	543	4.30	4.35	0.93
AD	926	4.40	3.85	0.18
AF	540	4.60	0.95	12.28
AF	812	5.10	6.35	13.54
AF	805	5.40	3.19	10.25
AE	866	5.70	19.93	2.22
AC	312	5.70	382.00	ND

TABLE 1: NEW BEDFORD HARBOR SEDIMENT ORGANIC DATA
1984 to 1986
(Continued)

		TOTAL PCBs	TOTAL PAHs	TOTAL PHTHALATES
* SAMPLE NUMBER		(mg/kg)	(mg/kg)	(mg/kg)
AD	565	5.80	1.43	2.29
AD	594	6.81	ND	ND
AD	930	6.95	49.10	0.83
AF	815	7.00	6.88	16.65
AD	578	7.20	5.54	1.26
AD	831	7.43	ND	ND
AD	836	8.51	ND	ND
AD	580	11.20	ND	8.29
AD	927	11.60	13.67	3.00
AF	228	11.80	8.37	4.50
AD	924	13.50	64.25	4.50
AD	837	13.80	2.48	ND
AC	316	14.00	9.51	2.50
AD	567	16.00	0.68	2.75
AF	811	16.70	6.17	12.14
AF	804	17.00	40.67	9.01
AC	323	17.20	46.70	5.70
AF	232	20.80	7.33	4.40
AD	575	20.90	9.80	6.49
AD	943	21.70	ND	ND
AD	922	21.70	9.50	1.66
AD	941	22.50	5.40	1.22
AD	559	25.60	10.49	6.06
AE	872	27.00	24.26	11.74
AD	921	28.40	9.82	0.63
AD	833	29.40	ND	ND
AF	803	30.60	7.98	8.55
AD	932	33.00	27.86	8.20
AD	570	39.00	6.29	1.46
AD	834	40.80	ND	0.42
AD	583	45.00	0.58	6.19
AD	599	49.40	7.86	ND
AF	806	59.00	6.90	11.33
AD	586	60.00	0.06	ND
AD	579	66.00	15.40	0.28
AD	562	67.00	14.73	4.12
AD	574	96.00	5.25	0.17
AD	584	104.00	3.79	2.61
AD	827	121.00	45.74	1.52
AD	592	161.00	21.40	ND
AD	934	218.00	20.10	76.00
AD	826	270.00	4.27	2.61
AD	931	330.00	291.00	2.20
AC	307	430.00	235.00	38.40
AD	582	440.00	30.86	7.83
AD	585	500.00	17.66	ND

TABLE 1: NEW BEDFORD HARBOR SEDIMENT ORGANIC DATA
1984 to 1986
(Continued)

		TOTAL PCBs	TOTAL PAHs	TOTAL PHTHALATES
* SAMPLE NUMBER		(mg/kg)	(mg/kg)	(mg/kg)
AD	595	550.00	13.26	ND
AD	835	1140.00	ND	9.43
AD	933	1260.00	487.10	69.00
AD	937	1620.00	ND	0.78
AD	942	1640.00	33.30	23.00
AD	576	1900.00	91.60	26.00
AD	830	2090.00	22.04	12.74
AC	337	2150.00	927.00	36.00
AD	825	2250.00	ND	17.80
AD	832	2650.00	18.56	12.96
AC	327	9800.00	410.00	3800.00

* = AC Sample Numbers correspond to the Battelle Hot Spot Sampling Program.

AD Sample Numbers correspond to the US Army Corps of Engineer Wetlands and FIT Sampling Program.

AE Sample Numbers correspond to the NUS/GZA Harbor Grid Sampling Program.

AF Sample Numbers correspond to the NUS/GZA Harbor Grid Sampling Program.

Total PAH concentrations represent the sum of: acenaphthene, acenaphthylene, anthracene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, benzo(k)fluoranthene, benzo(g,h,i)perylene, chrysene, dibenzo(a,h)anthracene, fluoranthene, flourene, indeno(1,2,3-cd)pyrene, naphthalene, 2-methylnaphthalene, 2-chloronaphthalene, phenanthrene, and pyrene. Total phthalate ester concentrations represent the sum of: bis(2-ethylhexyl)phthalate, di-n-octyl phthalate, di-ethyl phthalate, butyl benzyl phthalate and dimethyl phthalate. Note: Di-n-butyl phthalate concentrations are included in the total calculation; however, this compound was not validated in all data packets (see NUS Data Packet Case No. 5450, April 28, 1986).

sample locations screened out were done so based on the assumption that reducing the PCB sediment concentration during remediation will effectively reduce the associated PAH and phthalate ester concentrations.

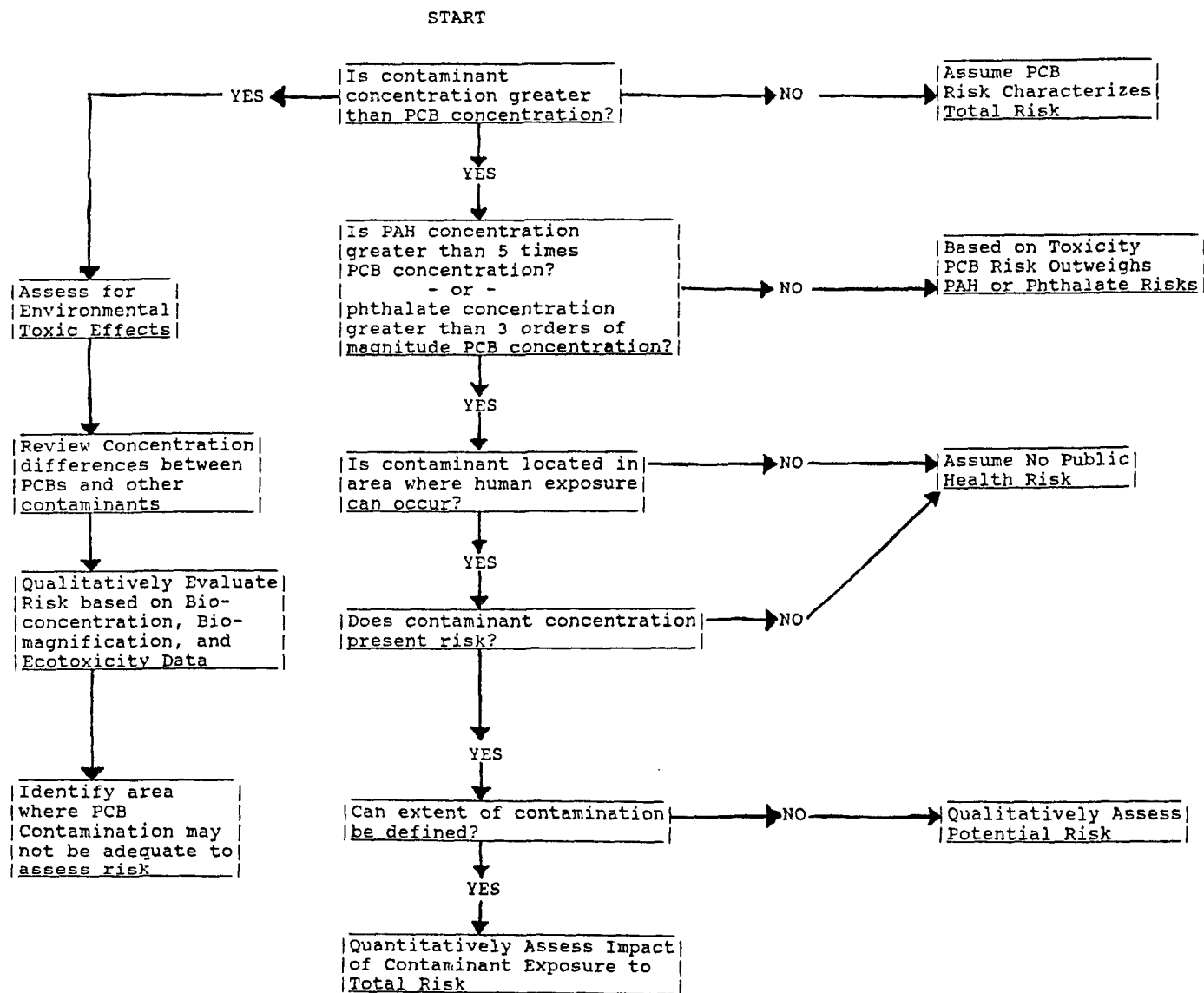
Sixty-nine sample locations fit the above criteria, 43 sample locations had elevated PAH concentrations and 55 sample locations had elevated phthalate ester concentrations relative to the corresponding PCB concentrations. Thirty sample locations had both elevated PAH and phthalate ester concentrations.

In addition to the above criteria, sample locations where low levels (i.e., below 1 ppm) of both PCBs and PAHs and/or phthalate ester compounds were detected were evaluated to determine the significance of the incremental risks associated with low level contaminant exposure. Direct contact exposure to these sediments and the bioconcentration and biomagnification potential of these compounds were evaluated as part of this review. Five sample locations were identified as having low level contamination. PAHs and PCBs were the only contaminants detected at these locations. ~~The percent contribution of PAH contamination to the total contamination ranged from 11 to 28 percent, indicating that PCBs constitute the majority of the contamination at these sample locations.~~ Based on this review, Jordan believes the risks associated with exposure to sediments in these areas will be related to the level of PCB contamination.

2.2.2 Phase II Screening

The focus of the second phase of the review process was to qualitatively evaluate the potential risks associated with the contamination detected at locations where PAH and/or phthalate ester contamination exceeded the PCB contamination. This involved assessing both the toxicity of, and potential exposure to, PAHs and phthalate ester compounds. Section 3.0 describes separately the evaluation process for determining the potential risks to human health and the environment from PAH and phthalate ester contamination. An outline of this review process is presented in Figure 1. It should be noted that the intent of Jordan's analysis was not to perform a detailed risk characterization for PAH and phthalate contamination. Instead, toxicity and exposure information is used to qualitatively assess comparative risks posed by the various contaminants to determine if the risk assessment should address the potential risks from other contaminants.

FIGURE 1
FLOW DIAGRAM OF REVIEW PROCESS



3.0 DISCUSSION AND RESULTS

3.1 PAHs: PUBLIC HEALTH EVALUATION

PAHs were detected in greater concentrations than PCBs at 43 sampling locations. The difference between total PAHs and total PCBs concentration ranged from 0.12 to 376 ppm (Table 2). Because of the greater PAH concentration at these locations, it can not be assumed that a risk assessment based only on PCB concentrations can adequately characterize the risks from exposure to these sediments. This assumption can be supported or refuted only after reviewing both the toxicity of and exposure to PAH compounds with respect to PCB compounds. The first review involves assessing PAH toxicity relative to PCB toxicity to determine when, or if, it is appropriate to characterize the overall risks based only on PCBs contamination.

3.1.1 Toxicological Evaluation

As classes of compounds, both PCBs and PAHs are suspect human carcinogens with B2 classifications from the EPA. Both PCBs and PAHs refer to a class of compounds which exhibit varying degrees of toxicity. The carcinogenic and non-carcinogenic toxic potential of PCB compounds are related to certain isomer groups, but because PCBs or Aroclors constitute a mixture of isomers, PCBs as a class of compounds are considered to be carcinogenic. Certain individual PAH compounds have demonstrated a carcinogenic potential by various routes of exposure while others are considered to be noncarcinogenic or have not been tested extensively. Since individuals are expected to be exposed to a complex mixture with varying PAH constituents, PAHs are also considered to be carcinogenic.

Since the carcinogenic risks often drive the risk assessment and remediation processes, it is appropriate to focus this analysis on the carcinogenic potential of these compounds. A value of 11.53 (mg/kg-day)⁻¹ has been derived by the Carcinogenic Assessment Group (CAG) based upon the data from a single PAH compound, benzo(a)pyrene, which is the most potent carcinogen of those PAHs tested for carcinogenicity. For PCBs, a carcinogenic potency factor of 4.34 (mg/kg-day)⁻¹ has been developed by the CAG based on the testing done with Aroclor 1260. The toxicity of both PCBs and PAHs, as described by these CAG values, is based on conservative assumptions.

The carcinogenic potency values for these two groups of compounds (11.53 and 4.34 (mg/kg-day)⁻¹) are within one order of magnitude, indicating that PAHs and PCBs are thought to exhibit the same relative carcinogenic potential. This assumption is supported by the relative potency index values for these compounds which are calculated by multiplying the unit cancer risk by the molecular weight of the compound. This index

TABLE 2: SEDIMENT SAMPLES WHERE TOTAL PAH CONCENTRATIONS
EQUAL OR EXCEED CORRESPONDING PCB CONCENTRATIONS
1984 TO 1986

		SAMPLE LOCATION	TOTAL PCB	TOTAL PAH	PAH - PCB
* SAMPLE NUMBER			(mg/kg)	(mg/kg)	(mg/kg)
AD	927	Estuary	11.60	13.67	2.07
AF	812	Harbor	5.10	6.35	1.25
AF	807	Harbor	1.00	1.43	0.43
AF	810	Harbor	0.59	0.94	0.35
AF	802	Harbor	3.20	6.68	3.48
AE	823	Harbor	0.24	0.52	0.28
AF	538	Harbor	0.64	1.47	0.83
AF	804	Harbor	17.00	40.67	23.67
AD	563	Estuary	0.07	0.19	0.12
AC	323	Estuary	17.20	46.70	29.50
AF	813	Harbor	2.40	7.19	4.79
AF	229	Harbor	0.66	2.11	1.45
AD	581	Estuary	3.10	10.58	7.48
AE	866	Harbor	5.70	19.93	14.23
AD	828	Estuary	1.48	7.00	5.52
AD	924	Estuary	13.50	64.25	50.75
AF	227	Harbor	3.49	17.95	14.46
AF	801	Harbor	0.74	4.01	3.27
AF	816	Harbor	0.80	4.72	3.92
AD	596	Estuary	0.24	1.62	1.38
AD	930	Estuary	6.95	49.10	42.15
AD	598	Estuary	1.50	11.10	9.60
AD	569	Estuary	0.04	0.34	0.30
AF	541	Harbor	0.52	5.93	5.41
AF	814	Harbor	0.59	9.16	8.57
AF	176	Harbor	1.10	21.26	20.16
AC	312	Estuary	5.70	382.00	376.30
AF	230	Harbor	ND	0.43	0.43
AF	231	Harbor	ND	0.12	0.12
AC	330	Estuary	ND	1.91	1.91
AC	333	Estuary	ND	2.74	2.74
AF	542	Harbor	ND	0.13	0.13
AD	556	Estuary	ND	0.80	0.80
AD	558	Estuary	ND	1.10	1.10
AD	560	Estuary	ND	0.20	0.20
AD	561	Estuary	ND	0.20	0.20
AD	564	Estuary	ND	0.20	0.20
AD	572	Estuary	ND	0.04	0.04
AD	573	Estuary	ND	1.60	1.60
AD	588	Estuary	ND	0.69	0.69
AE	821	Harbor	ND	0.10	0.10
AE	867	Harbor	ND	5.82	5.82
AE	870	Harbor	ND	0.12	0.12

TABLE 2: SEDIMENT SAMPLES WHERE TOTAL PAH CONCENTRATIONS
EQUAL OR EXCEED CORRESPONDING PCB CONCENTRATIONS
1984 TO 1986

* = AC Sample Numbers correspond to the Battelle
Hot Spot Sampling Program.

AD Sample Numbers correspond to the US Army Corps
of Engineer Wetlands and FIT Sampling Program.

AE Sample Numbers correspond to the NUS/GZA
Harbor Grid Sampling Program.

AF Sample Numbers correspond to the NUS/GZA
Harbor Grid Sampling Program.

is used to compare the potency of different carcinogens. PCBs have a relative potency index of $1 \times 10^{+3}$ and PAHs have a value of $3 \times 10^{+3}$. Using these values to compare the relative potency (on a per mole basis) of these compounds, it appears that under similar exposure conditions (i.e., similar exposure concentrations) and assumptions (i.e., absorption rates), the carcinogenic risks for these two compounds will be within the same order of magnitude. Numerically, the risk based on only PCB contamination will be approximately one-third the risk based on PAH compounds (i.e., 1.3×10^{-6} vs. 3.9×10^{-6}). Given the uncertainty associated with deriving numerical risk estimates, it is Jordan's opinion that a difference of less than an order of magnitude (a factor of ten) can be considered insignificant.

Remedial alternatives are often evaluated based on attaining total carcinogenic risks within the range of 10^{-4} to 10^{-7} . Therefore, for conditions where the PCB and PAH concentrations are similar, a risk estimate based only on PCB contamination will provide an adequate estimate of the overall risks associated with concurrent exposure to PCBs and PAH compounds. The risk estimates based only on PCB contamination will be within a factor of 3 of the risk based on PAH and PCB contamination. Developing remedial alternatives aimed at decreasing the estimated PCB risks should also effectively reduce the risks from exposure to PAHs.

For areas where the concentrations of PCBs and PAHs are not similar, a separate risk assessment may be necessary to accurately reflect the overall risks from contaminant exposure. To identify these areas, locations where the PAH concentrations exceeded the corresponding PCB concentration by more than a factor of five (one-half an order of magnitude) were identified. This concentration difference is great enough such that the risk estimate developed for PAHs would be close to one order of magnitude larger than the risk estimate developed for PCBs.

3.1.2 Exposure Evaluation

Sixteen sample locations were screened out because the concentration difference between PCBs and PAHs were within a factor of five. Twenty-five locations showed PAH concentrations at levels greater than five times the corresponding PCB concentrations and, as such, could significantly contribute to the overall risks from contaminant exposure. To determine the significance of these PAH concentrations, the 25 sampling locations were evaluated based on exposure considerations such as location within the estuary and harbor and ease of access to these sediments. This provided information on the type and extent of exposure which could result in contact with contaminated sediments.

Jordan's evaluation identified the western shoreline of the harbor and estuary and the eastern wetlands in the estuary as areas where exposure to contaminated sediments was likely (Figures 2 and 3). Areas of high PAH concentrations detected in the middle of the harbor and estuary are not expected to contribute to the overall risk, as human exposure in these areas is expected to be minimal.

Eighteen sample locations are situated in areas along the western and eastern wetlands shorelines. Twelve are located in the wetlands along the eastern shore of the estuary, four are located near the western shore (three north of the Coggeshall Street Bridge, and two near the Aerovox Plant), and two are located in the harbor area. The potential risks associated with exposure to PAHs in these three areas will be evaluated separately due to the differences in observed concentrations and exposure considerations.

3.1.2.1 Estuary Area

The wetland area along the eastern shore of the estuary shows the most widespread distribution of elevated levels of PAHs with respect to the corresponding PCB concentrations. The PAH concentrations range from 0.04 to 11.1 ppm (mean of 1.6 ppm) and the corresponding PCB concentrations range from Not Detected (ND) to 1.5 ppm.

To determine the relative magnitude of risk associated with exposure to sediments contaminated with PAHs at these observed concentrations, preliminary risk estimates were developed using the following exposure assumptions: 35 kg child is exposed to 10 grams of sediment per contact with 25 contacts per year for 10 years out of a 70-year lifetime with dermal absorption rate of 5 percent and a unit cancer risk of 11.53 (mg/kg-day)⁻¹ (the unit cancer risk derived based on the toxicity of benzo(a)pyrene). The risks associated with exposure to PAHs at the mean concentration of 1.6 ppm is 2.6×10^{-6} and for exposure to the maximum concentration of 11.1 is 1.8×10^{-4} . These carcinogenic risks fall within the target range of 10^{-4} to 10^{-7} incremental carcinogenic risks used to develop and evaluate remedial alternatives. Based on these calculations, although some risk is associated with PAH exposure in the wetland area, the risk is not greater than 10^{-4} . If a reduction in this risk is considered necessary, remedial actions would be required in this area. The risks associated with PCB exposure in this same area range from 8.5×10^{-8} to 1×10^{-6} . These risks also fall within or below the target range of 10^{-4} to 10^{-7} .

The total carcinogenic risk resulting from concurrent exposure to PCBs and PAHs can be obtained by summing the individual carcinogenic risks. The total carcinogenic risk resulting from

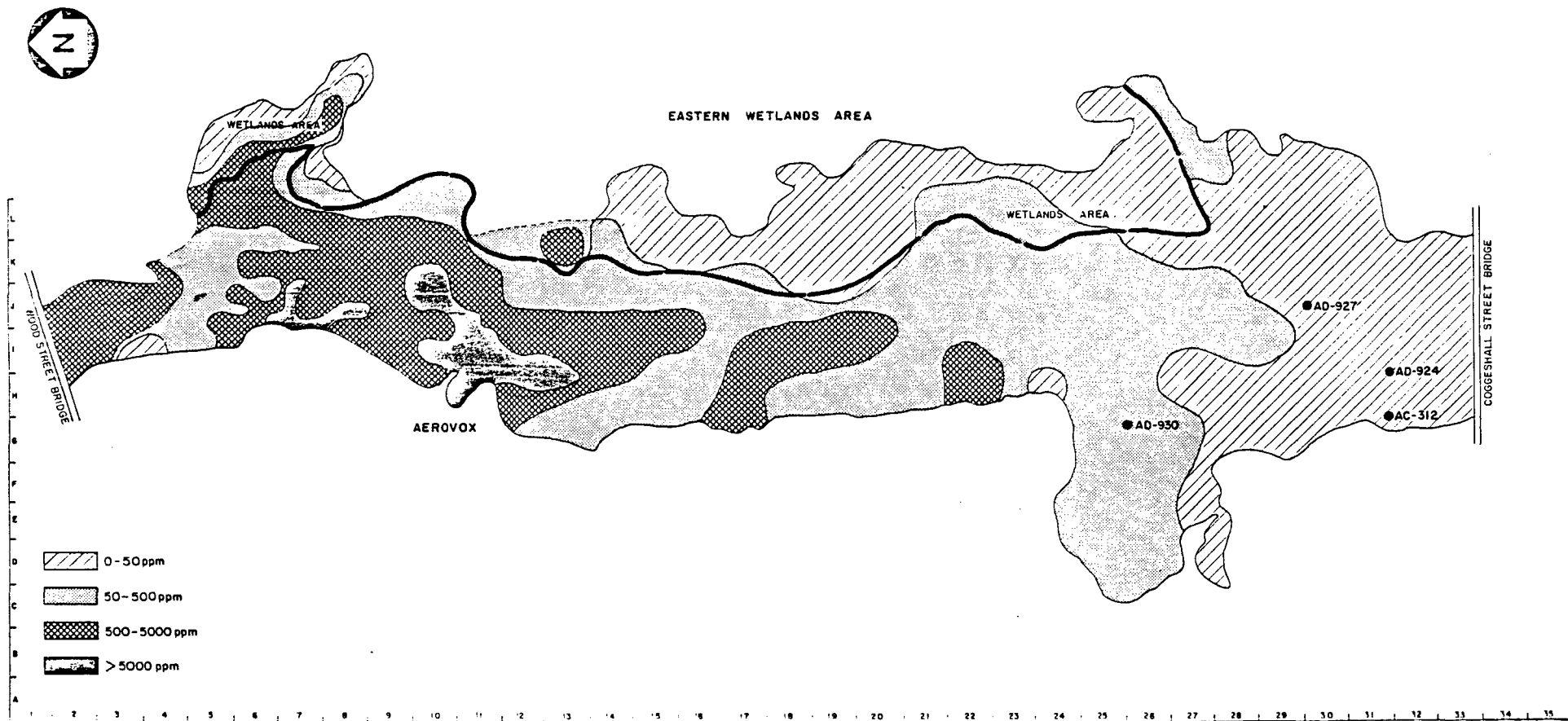
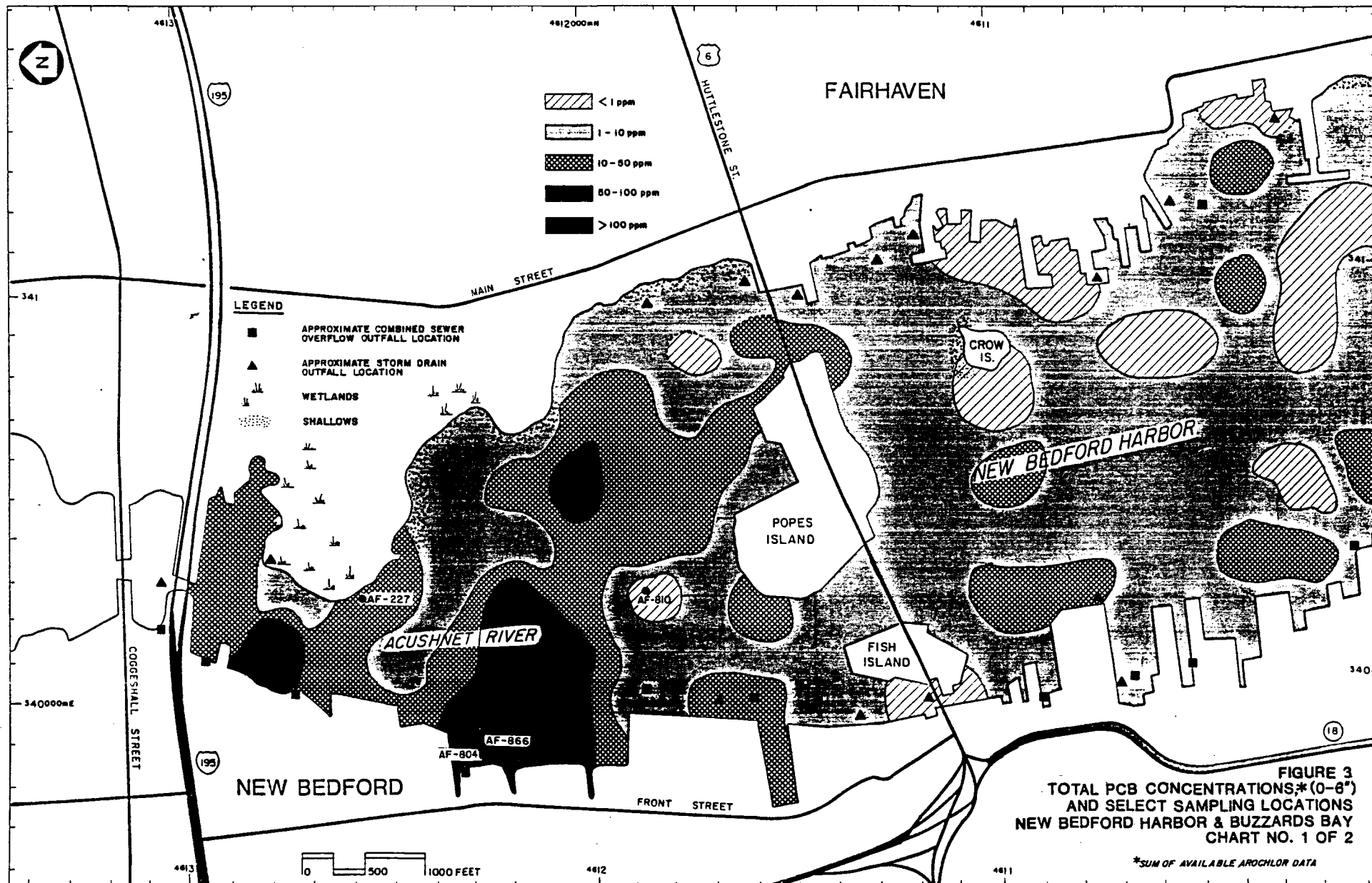


FIGURE 2
TOTAL PCB CONCENTRATIONS*(0-12")
AND SELECT SAMPLING LOCATIONS
NEW BEDFORD HARBOR
ACUSHNET RIVER ESTUARY

*SUM OF AVAILABLE AROCHLOR DATA



exposure to the mean concentrations of PCBs and PAHs is 2.6×10^{-6} and 1.9×10^{-5} for exposure to the maximum concentrations of PCBs and PAHs. As with the individual carcinogenic risks, these total risk estimates also fall within the 10^{-7} to 10^{-4} target range.

The sample locations on the western shoreline of the estuary are more widespread, two being located near the Aerovox Plant and three located south of the plant near the Coggeshall Street Bridge. The samples collected from locations near the Aerovox Plant were taken at depths of 36 inches and had PAH concentrations of 1.91 and 2.74 ppm. While the PCB concentrations at these same locations were both ND, the PCB concentrations detected in surface sediments around this area ranged from 50 to 500 ppm. Because the PAH concentrations are well below the range of PCBs associated with this area, the risks associated with exposure to PCBs will outweigh the risks attributed to PAH exposure. Therefore, risk estimates based only on PCB contamination will be adequate to characterize the risks associated with sediment exposure in this area.

Of the three samples taken from the western shore north of the Coggeshall Street Bridge, one (AD 930) was located in an area where the PCB concentration ranged from 50 to 500 ppm. At sample location AD 930 the measured PCB concentration was 7 ppm at a depth of 6 to 12 inches, while the concentration of PAHs at this depth was 49 ppm (PAH concentrations were not measured in the other sample locations.) The concentrations of PCBs other than at this location and depth are high enough that the risks associated with exposure to sediments will be driven by the presence of PCBs.

At sample locations AC 312 and AD 924, the PAH concentration exceeded the associated PCB concentration by close to a factor of eight and a factor of five, respectively. Therefore, PCBs and PAHs can not be considered to be present at similar concentrations in these areas, suggesting that the risks may be significantly increased by the presence of PAH compounds. The incremental carcinogenic risks associated with exposure to the average PAH concentration at these two locations is 3.7×10^{-4} , while the incremental carcinogenic risk associated with PCB exposure at these locations is 5.8×10^{-6} . These risk estimates indicate that the overall risk from exposure to sediments in this area may be underestimated by only considering PCBs. Therefore, PAH compounds should be considered when assessing exposure and risk in this area (see Figure 2).

3.1.2.2 Harbor Area

The majority of samples within the harbor area (south of the Coggeshall Street Bridge to the Hurricane Barrier) with elevated levels of PAHs were taken at depths ranging from 6 to 18 inches

with only a few located near the shoreline. These two factors (depth and location) suggest that exposure to PAH contaminated sediments in the harbor area will be limited. One surface sample (AF 227), taken on the eastern shore south of the Coggeshall Street Bridge, had measured PAH concentrations of 18 ppm (PCB concentration is 3.5 ppm) (see Figure 3). No other locations near AF 227 were sampled for HSL compounds. Given the high concentration of PAHs detected in this sample, the risks associated with exposure may be underestimated by addressing only PCB contamination. Because of the limited data, it is impossible to estimate the extent of PAH contamination in this area; however, this sample is located approximately 1,500 feet south of the Coggeshall Street Bridge and approximately 2,000 feet south of the area in the estuary showing elevated PAH concentrations. It is not possible to determine if the contamination detected in the harbor is part of the PAH contamination seen in the southern portion of the estuary.

3.1.3 Summary

Jordan's review of PAH and PCB distribution identified the southwestern shoreline of the estuary as being contaminated with PAHs at levels which may significantly contribute to the overall risks associated with sediment exposure. When assessing the risks associated with direct contact exposure in this area, the contribution from PAH contamination should be included. Currently, it is not possible to determine the extent of this contamination and therefore identify areas where the risks may be underestimated.

3.2 PAHS: ENVIRONMENTAL EVALUATION

The public health evaluation identified the southwestern shoreline of the estuary as being contaminated with PAHs at levels which may significantly contribute to the overall risks associated with sediment exposure. Based on this review, it was determined that a risk evaluation based on PCBs was not sufficient to characterize the overall risks to public health at New Bedford Harbor. In addition to public health concerns, an evaluation of the potential environmental impacts of PAH contamination must be performed to determine if a risk assessment based on PCBs alone is adequate to characterize the potential risks to the aquatic ecosystem. If the additional environmental risks from exposure to PAHs are significant, then PAHs should be included as part of the risk assessment for New Bedford Harbor.

3.2.1 Toxicological Evaluation

The limited amount of ecotoxicity data for total PAHs or any of the individual PAH compounds, and the absence of Ambient Water Quality Criteria (AWQC) for these compounds make it difficult to assess the potential impacts of contaminant exposure on the

aquatic ecosystem. In the absence of toxicity data or sediment criteria, Jordan compared bioconcentration/biomagnification (uptake and retention) data for PAHs and PCBs to determine the relative contribution of these contaminants to the expected body burdens in aquatic organisms at New Bedford Harbor. This information is used to identify areas in New Bedford Harbor where PAHs contamination may contribute significantly to the overall body burden of contaminants in fish tissue. While this information cannot specifically be used to evaluate the relative toxicity or risk of PCBs and PAHs, it does provide a comparison of the uptake and retention of these compounds in aquatic organisms.

In addition to the use of bioconcentration data, some limited information comparing PCB and PAH contamination in sediments is available. Chapman (1986) conducted a study in Puget Sound, Washington, using comparative data for sediment chemistry, sediment bioassays, and bottom fish histopathology to derive quantitative site-specific sediment criteria for three chemical contaminants (lead, PAHs, and PCBs). The criteria, in terms of concentrations at or below which biological effects were shown to be minimal, were (dry weight sediment): lead, 50 ug/g (ppm); combustion polyaromatic hydrocarbons, 3.8 ug/g; and total polychlorinated biphenyls, 0.1 ug/g (Chapman, 1986). Although these sediment quality criteria are site-specific to Puget Sound, and are directly applicable only when all three chemicals occur in the same location, they can still offer some understanding of the potential toxicity of sediment-bound PAH and PCB compounds to aquatic life. Jordan used Chapman's (1986) data as a screening tool to identify areas within New Bedford Harbor where the PAH concentrations may significantly contribute to the overall toxicity of contaminated sediments to aquatic life.

The following discussion uses the bioconcentration data and Chapman's sediment criteria values to assess the potential toxicity of sediment-bound PAHs and PCBs in New Bedford Harbor.

Bioconcentration factors (BCFs) for PAHs in marine aquatic organisms appear to be much lower than those measured for PCBs. Studies conducted by Reichert (1985) using the amphipods Eohaustorius washingtonius and Rhepoxynius abronius showed accumulated levels of benzo(a)pyrene (BaP)-derived radioactivity after one day of exposure to sediments containing [H] BaP. While Reichert (1985) concluded that amphipods do bioconcentrate BaP, no BCF were developed or were available in the literature for this species. Studies conducted with the phylogenetically similar amphipod Pontiporeia afinis exposed to PCBs showed a BCF of 1709. Because amphipods are a primary food source for higher trophic organisms, the bioconcentration process may result in biomagnification of contaminants up the food chain. The Eastern oyster (Crassostrea virginica) exhibited a BCF of 242 when

exposed to the PAH benzo(a)pyrene (EPA, 1980a). However, when exposed to the PCB Aroclor 1254, the Eastern oyster exhibited a BCF of 101,000 (EPA, 1980b). The BCFs for the tidepool sculpin (a bottom-dwelling marine fish) was observed to be 0.13 for PAHs and 84,000 for PCBs (EPA, 1980a). These studies indicate that the uptake of PCBs in marine fish is significantly greater than PAHs (in particular, BaP). In addition, a study conducted by Hansen (1986) showed that fish exposed to PAH-contaminated sediments (up to 169 mg/kg) from New Bedford Harbor had non-detectable levels (<10 ppb) of PAHs in their tissue. Hansen (1984) attributed these results to the fact that fish are highly efficient at metabolizing PAHs.

Based on these studies, it appears that aquatic organisms do not bioconcentrate PAHs to the same extent as PCBs. Therefore, biomagnification of PAHs up the food chain is not expected. PCBs however have been shown to bioconcentrate and biomagnify in marine organisms. This is attributed to PCBs' lipophilic characteristics and the fact that they are not metabolized by aquatic organisms to any great extent (EPA, 1980a). PCBs are expected to accumulate to a greater extent than PAHs in aquatic organisms.

The adverse effects of PCB or PAH body burdens on aquatic organisms are not well documented, making it difficult to determine the potential toxic effects to aquatic organisms from exposure to either PAH-or PCB-contaminated sediments. However, because PCBs have a greater propensity to bioconcentrate and biomagnify in aquatic organisms than PAHs, tissue concentrations of PCBs in aquatic organisms will be greater than PAHs when exposures are equal. Irrespective of the potential impacts to aquatic life, the potential for human exposure through ingestion of seafood will be greater for PCBs than for PAHs.

Chapman (1986) and other investigators have determined that chemical concentrations in sediments are often many times higher than water column concentrations. However, almost all toxicological criteria developed to date for protection of the aquatic environment are based on aqueous chemical concentrations. Chapman (1986) developed sediment quality criteria for PCBs, PAHs, and lead (Pb) for the protection of aquatic life, based on the results of sediment bioassays, with the amphipod, the oligochaete, and the English sole. Toxicity was observed at >3.8 ppm PAHs, >0.1 ppm PCBs, and >50 ppm Pb sediment concentrations, (dry weight). The observed toxic effects included acute lethality, respiratory rate elevation and depression, and liver lesions. Because of the possible interactions between PCBs, PAHs, and Pb, Chapman was unable to determine a no-effect level for a specific contaminant. Thus, strictly speaking, Chapman's criteria for PAH are applicable only if Pb and PCB levels do not exceed their associated criteria concentrations. In areas of New Bedford Harbor where these chemicals are collocated, the toxicological effect

concentrations derived by Chapman (1986) can be compared to observed sediment concentrations. Although no lead sediment concentrations are available for this evaluation, some extrapolation as to the potential toxicity of PAHs and PCBs in sediment can still be made.

3.2.2 Exposure Evaluation

In the following discussion, PCB and PAH sediment data are evaluated with respect to Chapman's (1986) data and bioconcentration/biomagnification studies to identify areas where the actual risks to aquatic organisms may be greater than the risk based only on PCBs.

The following analysis addresses the estuary and harbor areas separately. As in the public health exposure evaluation, this analysis focuses on sample locations where total PAH concentrations exceed the corresponding PCB concentrations. Table 2 is a list of sample locations evaluated for this analysis.

3.2.2.1 Estuary Area

Twenty-one sample locations within the estuary area contained PAH concentrations in excess of the corresponding PCB concentrations. Of these, 11 were collected within the wetland area on the eastern shoreline and one sample collected on the western shoreline near the Aerovox Plant. The concentration of PAHs at these locations ranged from 0.20 to 2.74 ppm. The corresponding PCB concentrations ranged from below the detection limit to 0.07 ppm.

Based on the Chapman (1986) study, toxicological effects to aquatic organisms are not expected to occur at sediment exposure concentrations below 0.1 ppm PCB and 3.8 ppm PAH. The observed PAH and PCB concentrations at these 12 locations fall below the criteria values observed by Chapman (1986), suggesting that the risks to aquatic organisms in these areas will be minimal.

Three additional samples collected from the wetland area on the eastern shoreline show levels of PAHs and/or PCBs in excess of Chapman (1986) 3.8 ppm and 0.1 ppm criteria values. These sample locations occur at the edge of the wetland area, two in or near the channel (sample AD 581 at the northern edge of the wetland and AD 598 at the southern edge of the wetland) and one (AD 596) located at the southern edge of the wetlands area. All three locations are near areas showing much higher concentrations of PCBs (30 to 70 ppm) suggesting that these locations may represent the transition area between high PCB contamination (channel) and low PCB contamination (eastern shoreline wetlands).

Based on the available toxicity data for PAH and PCB compounds, it is difficult to determine the relative risks posed by the concurrent exposure to PCBs and PAHs at these three locations. As stated, the Chapman (1986) study does not provide a no-effect sediment level for any one particular contaminant but rather a triad criteria value for PAHs, PCBs, and Pb. Without chemical-specific criteria values, it is not possible to determine which contaminant, PAHs, or PCBs will be the driving force of toxicity. At sample locations AD 581 and AD 598, both PCBs and PAHs occur in excess of the suggested criteria values, making it difficult to determine if a risk assessment based only on PCBs will adequately characterize the risk from exposure to both contaminants. However, as stated above, these two samples are located in a transition area of contamination surrounded by an area with substantially higher levels of PCBs. At location AD 596, PAH concentrations exceed the corresponding PCB concentration; however, only PCBs exceed the criteria value (PAH concentration is 1.62 ppm). On this basis, it appears that the risks to aquatic organisms may be adequately defined based on the area's overall higher PCB concentrations.

Two of the remaining six samples collected from the estuary showing elevated levels of PAHs and PCBs (AD 373 and AD 828) occur in areas located near highly contaminated PCB sediments (50 to 500 ppm). Both these samples were collected on the western shoreline directly north of the Aerovox Plant. Although the PAH concentrations exceed the 3.8 ppm value observed by Chapman (46.7 and 7 ppm), the PCB concentrations in this area are substantially higher than the PAH concentrations (50 to 500 ppm). The high concentrations suggest that PCBs will drive the risk assessment for this area. In addition, the tendency of aquatic organisms to bioconcentrate and biomagnify PCBs more readily than PAHs, suggests a potential increased risk to aquatic organisms from additional body burden of PCBs. Therefore, it is assumed that a risk assessment based only on PCB contaminant levels will adequately address the risks from PAH exposure.

Three samples (AD 927, AD 924, and AC 312 [see Figure 2]) in the estuary are located directly north of the Coggeshall Street Bridge. The PAH concentrations at these locations are 13.67, 64.25, and 38.2 ppm, while the PCB concentrations range from 4.7 to 13.5 ppm. The concentrations of PCBs and PAHs at all three locations exceed Chapman's criteria concentrations, indicating a potential risk to aquatic organisms from exposure to these sediments. At one location (AD 930), PAH concentrations exceed PCB concentrations in the depth range of 6 to 12 inches. No toxicity data are available to assess the contribution of either contaminant to the total risk. However, given the greater PAH concentrations detected at the three areas with respect to PCB concentrations, it appears that a risk assessment based only on PCBs may underestimate the total risk to aquatic organisms. In addition, this section of the estuary was identified in the

Public Health evaluation of PAH contamination as an area which may significantly contribute to the overall risk to human health. It appears that the risk to aquatic organisms may also be impacted by the presence of PAH compounds, and as such, this area should be evaluated based on both PCB and PAH contamination.

3.2.2.2 Harbor Area

Twenty-two samples taken from the harbor and bay area (south of the Coggeshall Street Bridge beyond the Hurricane Barrier) showed PAH concentrations in excess of the corresponding PCB concentrations. For purposes of this discussion, this lower portion of the Acushnet River is divided into sections to focus the evaluation of PAH and PCB contamination. Reference is made to Table 2 for sample identification and corresponding PCB and PAH concentrations, and Figure 3 for approximate sample locations.

Six sample locations, collected between the Coggeshall Street Bridge and Popes Island, show PAH concentrations to be in excess of the corresponding PCB concentrations. At two locations, the PAH levels were 0.12 and 0.43 ppm with the corresponding PCB concentrations below the detection limit. Because both the PAH and PCB concentrations at these locations are below Chapman's criteria values of 3.8 ppm and 0.1 ppm, respectively, minimal risk to aquatic organisms is expected.

At the remaining four locations, the PAH concentrations are in excess of PCB concentrations and range from 0.94 to 40.67 ppm. The corresponding PCB concentrations range from 0.59 to 17.00 ppm. Since both contaminants exceed Chapman's criteria value of 0.1 ppm PCB and 3.8 ppm PAH, there may be some risk to aquatic life. However, given the limited toxicity data, it is not possible to determine which contaminant, PAHs or PCBs, will cause greater toxicity to aquatic organisms at these locations.

Currently, it is not possible to determine the extent of PAH contamination in the area defined by the four samples. Two of the four samples (AE-866 and AF-804) are located within the same general area, on the western shoreline midway between Coggeshall Street Bridge and Popes Island. Four combined sewer overflow (CSO) outfalls are also located in this area. The remaining two samples are located distant from this area, with AF-810 located in the middle of the channel and AF-227 located on the eastern shoreline. Because of the wide distribution of these four sample locations, it is difficult to determine how much of an area may be affected by PAH contamination. This, in addition to the limited ecotoxicity data available to assess PCB and PAH sediment contamination, make it impossible to determine if a risk assessment based only on PCB contamination will adequately characterize the risks in this area.

Eleven samples collected between Popes Island and the Hurricane Barrier had elevated PAH concentrations with respect to the corresponding PCB concentrations. At two locations, both the PCB and PAH concentrations are below Chapman's criteria concentrations of 0.1 ppm PCB and 3.8 ppm PAH, suggesting minimal adverse effects to the aquatic ecosystem. At the remaining nine sample locations, the PAH concentrations range from 1.47 to 21.26 ppm with PCB concentrations ranging from 0.52 to 5.10 ppm. The concentrations of both contaminants are, on the average, lower than those detected in other areas of the harbor/estuary with eight of the nine sample locations having concentrations of PCBs and PAHs less than 10 ppm. While it is not possible to determine which contaminant, PCBs or PAHs, will contribute more to the toxicity, the relatively low concentrations, coupled with the fact that PCBs bioconcentrate and biomagnify more readily than PAHs, suggests an additional risk to aquatic organisms from PCBs. Therefore, it is assumed that a risk assessment based only on PCB contaminant levels will adequately address the risk from concurrent exposure to PAHs.

Five samples taken along the western shoreline, south of the Hurricane Barrier (AF-542, AF-807, AF-802, AF-823, and AF-814) had PAH concentrations in excess of the corresponding PCB concentrations. The concentrations of PCBs and PAHs at one sample location were both below the Chapman criteria values, suggesting minimal toxicity to aquatic organisms at this location. The concentration of PAHs and PCBs at the remaining four locations ranged from 0.52 to 9.16 ppm and 0.24 to 3.20 ppm, respectively. The relatively low concentrations of both contaminants, coupled with the tendency for PCB to bioconcentrate and biomagnify in aquatic organisms, suggest that a risk assessment based only on PCBs will be sufficient to assess the risks to aquatic organisms within this area.

3.2.3 Summary

Based on the above evaluation, Jordan has identified many areas in New Bedford Harbor where the PAH concentration exceeds the corresponding PCB concentrations. The limited information available to assess the toxicity of either sediment-bound PCBs or PAHs precludes an assessment of which contaminant presents the greatest risk to aquatic organisms. However, using the three criteria values developed by Chapman, and bioconcentration/biomagnification data, Jordan was able to identify areas where the risks from PAH exposure were expected to be minimal and areas where the risks from PAH exposure were expected to be significant. One area within the estuary was identified as being contaminated with PAHs at levels which may significantly contribute to the overall risk from contaminant exposure. This area is located north of the Coggeshall Street Bridge and extends from the middle of the channel to the western shoreline. Based on the available data, a risk assessment for

only PCBs may underestimate the overall risk to aquatic organisms from the concurrent exposure to PAH and PCB compounds.

3.3 PHTHALATE ESTERS: PUBLIC HEALTH EVALUATION

Phthalate ester compounds were detected at levels greater than the corresponding PCB concentration at 56 sample locations. The concentration difference between total phthalate ester and PCB concentrations ranged from 0.04 to 91 ppm (Table 3). Because of the greater phthalate ester concentrations at these locations, it can not be assumed that a risk assessment based only on PCB contamination will adequately characterize the risk from concurrent exposure to phthalate ester compounds. To determine if additional risk estimates for phthalate ester exposure is necessary, Jordan reviewed the toxicity of phthalate compounds relative to PCBs. This information, combined with exposure considerations, provided an indication of the relative risks attributable to the presence of phthalate ester compounds in the New Bedford Harbor area.

3.3.1 Toxicological Evaluation

As previously stated, PCBs as a class of compounds are considered to be carcinogenic. The CAG of the EPA has derived a unit cancer factor of 4.34×10^{-3} (mg/kg-day)⁻¹ and a relative potency factor of 1×10^{-3} for these compounds. Of the six individual phthalate ester compounds comprising the total phthalate ester concentration only one, bis (2-ethylhexyl) phthalate (DEHP) has been shown to be carcinogenic. The unit cancer factor for DEHP is 6.84×10^{-4} (mg/kg-day)⁻¹ and the potency index value is 3×10^{-1} . All other phthalate ester compounds have been shown to exhibit a low order of toxicity. Repeated exposures to these phthalate esters, orally, dermally, and through inhalation, appear to produce few, if any, toxic effects in animals except at high exposure concentrations (Autian, 1973).

Using the potency index values for PCBs and DEHP (as a conservative representation of all phthalates) as an indication of the carcinogenic potential of these two compounds, it appears that PCBs are approximately four orders of magnitude more potent than phthalate ester compounds. Under similar exposure conditions (amount of sediment contacted) and assumptions (absorption rates), the phthalate ester concentrations would have to be four orders of magnitude greater than the corresponding PCB concentrations to present a similar risk. In situations where the phthalate concentrations are within four orders of magnitude of the PCB concentration, the risks from PCB exposure may outweigh the risk from phthalate exposure (assuming DEHP represents the majority of the phthalates detected).

TABLE 3: SEDIMENT SAMPLES WHERE TOTAL PHTHALATE CONCENTRATIONS
EQUAL OR EXCEED CORRESPONDING PCB CONCENTRATIONS
1984 TO 1986

		TOTAL PCB	TOTAL PHTHALATES	PHTH - PCB
* SAMPLE NUMBER		(mg/kg)	(mg/kg)	(mg/kg)
AF	539	3.86	4.57	0.71
AE	820	1.88	2.70	0.82
AF	227	3.49	5.60	2.11
AF	805	5.40	10.25	4.85
AE	869	4.20	8.80	4.60
AF	815	7.00	16.65	9.65
AD	563	0.07	0.17	0.10
AF	812	5.10	13.54	8.44
AF	540	4.60	12.28	7.68
AF	809	2.30	6.19	3.89
AD	566	1.10	3.20	2.10
AD	581	3.10	9.39	6.29
AF	813	2.40	8.84	6.44
AF	538	0.64	2.40	1.76
AD	571	0.18	0.74	0.56
AF	229	0.66	3.05	2.39
AE	871	0.83	3.94	3.11
AF	801	0.74	6.37	5.63
AE	823	0.24	3.20	2.96
AF	810	0.59	10.21	9.62
AF	814	0.59	10.76	10.17
AD	569	0.04	0.81	0.77
AF	541	0.52	10.87	10.35
AD	577	0.84	21.90	21.06
AD	557	0.39	12.25	11.86
AF	816	0.80	57.47	56.67
AF	176	1.10	92.00	90.90
AF	230	ND	0.45	0.45
AF	231	ND	0.64	0.64
AC	301	ND	0.47	0.47
AC	310	ND	1.10	1.10
AC	311	ND	1.50	1.50
AC	315	ND	1.10	1.10
AC	320	ND	0.76	0.76
AC	330	ND	1.80	1.80
AC	333	ND	1.60	1.60
AC	345	ND	15.84	15.84
AF	542	ND	1.29	1.29
AD	556	ND	27.65	27.65
AD	558	ND	18.53	18.53
AD	560	ND	0.19	0.19
AD	561	ND	0.18	0.18
AD	564	ND	0.15	0.15

TABLE 3: SEDIMENT SAMPLES WHERE TOTAL PHTHALATE CONCENTRATIONS
EQUAL OR EXCEED CORRESPONDING PCB CONCENTRATIONS
1984 TO 1986
(Continued)

		TOTAL PCB	TOTAL PHTHALATES	PHTH - PCB
* SAMPLE NUMBER		(mg/kg)	(mg/kg)	(mg/kg)
AD	572	ND	0.04	0.04
AD	573	ND	0.08	0.08
AE	822	ND	2.97	2.97
AE	824	ND	0.22	0.22
AD	839	ND	0.04	0.04
AE	867	ND	1.05	1.05
AE	868	ND	1.80	1.80
AE	870	ND	5.38	5.38
AD	919	ND	0.25	0.25
AD	928	ND	0.11	0.11

* = AC Sample Numbers correspond to the Battelle
Hot Spot Sampling Program.

AD Sample Numbers correspond to the US Army Corps
of Engineer Wetlands and FIT Sampling Program.

AE Sample Numbers correspond to the NUS/GZA
Harbor Grid Sampling Program.

AF Sample Numbers correspond to the NUS/GZA
Harbor Grid Sampling Program.

3.3.2 Exposure Evaluation

To provide a conservative review, the organic data were screened to identify areas where the phthalate concentrations were close to three orders of magnitude greater than the corresponding PCB concentrations. Phthalate concentrations in these areas were considered to present a potentially significant contribution to the overall risk. Only two locations met the above criteria; however, 29 samples had detectable phthalate concentrations with non-detectable PCB concentrations. These thirty-one samples were the focus of Jordan's evaluation.

The two sample locations with elevated phthalate concentrations relative to the PCB concentrations are located in the harbor area. Both are subsurface samples with phthalate concentrations of 57 and 92 ppm (corresponding PCB concentrations are 0.8 and 1.1 ppm). To provide a conservative estimate of the potential risks associated with exposure to phthalate esters, preliminary risk estimates were developed based on the same exposure assumptions used to evaluate PAH exposure (35 kg child exposed to 10 grams of sediment per contact with 25 contacts per year for a 10-year exposure duration and a dermal absorption factor of 5%). It was assumed that the total phthalate concentration was DEHP and that exposure to these subsurface sediments is likely. The risks associated with an exposure dose of 57 ppm is 5.5×10^{-5} and 8.8×10^{-5} for an exposure dose of 92 ppm. These risk estimates fall below the target range of 10^{-4} to 10^{-3} used in developing and assessing remedial alternatives and indicate that exposure to phthalate esters do not contribute significantly to the overall risks.

The risk estimates developed above indicate that exposure to phthalate compounds at concentrations up to 92 ppm do not result in a significant risk. In the areas where PCB concentrations were below the detection limit, the phthalate esters concentrations ranged from 0.04 to 28 ppm, based on the previous risk estimates, exposure to these sediments will not present a significant risk.

3.4 PHTHALATE ESTERS: ENVIRONMENTAL EVALUATION

The public health evaluation concluded that even under very conservative exposure assumptions, the risks from phthalate ester contamination will not be significant. In addition to public health concerns, an environmental evaluation was performed to determine the potential environmental impacts of phthalate ester contamination on aquatic organisms in order to determine if a risk assessment based on only PCBs can adequately characterize the additional environmental risks from exposure to phthalate compounds.

At 55 sample locations, phthalate esters were detected at levels greater than the corresponding PCB concentrations (Table 3). The concentration difference between total phthalate esters and PCB concentrations ranged from 0.04 to 91 ppm, with the percent difference ranging from 18 to 8,300 percent. Because of locations where phthalate ester concentrations exceed those of PCBs, it cannot be assumed that a risk assessment based only on PCBs can adequately characterize the additional risks from exposure to phthalates.

As in the case of PAHs, the limited amount of toxicity data for phthalate compounds, and the absence of Ambient Water Quality Criteria (AWQC), make it difficult to assess the potential impacts of phthalate ester compounds to the aquatic ecosystem. A study conducted by Group (1986) describes the environmental fate and aquatic toxicity of selected phthalate esters compounds. Jordan compared this data to PCB environmental fate and toxicity data to determine which compound may contribute more to the potential risks to aquatic organisms.

Group (1986) observed the EC50 (concentration at which 50 percent of the organisms tested showed a toxic effect) for daphnids and mysid shrimp exposed to water soluble diethyl phthalate (DEP) ranged from 0.8 to 120 mg/L (ppm). Similar tests conducted with daphnids and shrimp exposed to PCBs had an EC50 range of 1.3 to 3.0 ug/L (ppb). The EC50 concentration of these two compounds differ by more than an order of magnitude.

Group (1986) also observed that EC50 and LC50 (concentration at which 50 percent of the organisms tested exhibit mortality) values for C6-C13 side chain phthalate (Table 4) for both daphnids and mysid shrimp were "all greater than" the water solubility (0.1 to 1.2 mg/L) of these compounds. The toxicity of PCBs has been observed at concentrations well below its water solubility (20 ppb for Aroclor 1254).

Studies conducted by Group (1986) with phthalate esters show these compounds degrade readily, with half-lives of 1.9 to 27.7 days. The half-life for the phthalates tested averaged 8 days. Group (1986) concluded from his studies that phthalates are practically non-volatile and only sparingly soluble in water, and therefore do not pose a significant threat to the environment. Similar studies with PCBs show that these compounds are extremely persistent in the natural environment with very little degradation occurring (EPA, 1980a).

Although total sediment-bound phthalate ester concentrations exceed PCB concentrations at 55 sample locations, the low volatility and solubility of these compounds suggest that very

TABLE 4
 PHTHALATE ESTERS DETECTED AT NEW BEDFORD HARBOR
 USED IN EFFECTS STUDIES BY EDWARD GROUP (1986)

<u>Common Designation</u>	<u>Chemical Name</u>
DMP	Dimethyl phthalate
DEP	Diethyl phthalate
BBP	Butyl benzyl phthalate
610P	Di(n-hexyl, n-octyl, n-decyl) phthalate
DEHP	Di(2-ethylhexyl) phthalate

low concentrations of phthalate esters will be present in the water column. Experimental evidence indicates that:

- o although lower molecular weight phthalates (e.g., DEP) produced toxicological effects to aquatic organisms below maximum water solubility (0.8-12 mg/l), toxicity observed with PCBs occurs at much lower concentrations overall (1.3 to 3.0 ug/l) (EPA, 1980);
- o phthalate esters with side chains C6 or greater (e.g., DEHP) exhibit no toxicity to aquatic life up to their limits of water solubility;
- o PCBs are many times more persistent in the environment than phthalates; and
- o published literature studies for five representative phthalates have shown low to moderate potential for bioconcentration in aquatic life (Group, 1986). Similar studies with PCBs yielded bioconcentration factors greater than 670,000 (EPA, 1980a).

This data indicates that, when found in solution, phthalate esters are of very low toxicity to saltwater aquatic life. PCBs in solution are much more toxic to and bioconcentrate to a greater extent in aquatic organisms than phthalate ester compounds.

Jordan was unable to identify any applicable information describing the toxicity of sediment-bound phthalate esters to aquatic organisms; therefore, a quantitative analysis of the potential risks from sediment-bound phthalate compounds is not possible. However, given the low toxicity of water-soluble phthalate ester compounds and their relatively low bioconcentration/biomagnification potential (with respect to PCBs), phthalate ester compounds are not likely to present a significant risk to aquatic organisms in New Bedford Harbor.

3.5 EVALUATION OF COLLOCATED PCB, PAH, AND PHTHALATE ESTER COMPOUNDS

Thirty sample locations had PAH and phthalate ester concentrations in excess of the corresponding PCB concentrations. To assess the potential risks associated with concurrent exposure to all three contaminants requires an assessment of the relative toxicity of each compound class. The limited amount of ecotoxicity data, PCBs, PAHs, and phthalate ester compounds precludes an evaluation of the potential environmental risks associated with the concurrent exposure to all three compounds. Therefore, this evaluation focuses only on the potential public health risks.

PCBs and PAHs have about the same relative carcinogenic potential (as indicated by the potency index factors of 1×10^{-3} and 3×10^{-3} , respectively). Phthalate esters are much less potent, and only one phthalate compound (DEHP) has a potency index factor of 3×10^{-1} . Based on the relative potency, it appears that when these three compounds are collocated, the risk from PAH and/or PCB exposure will greatly exceed the risk from phthalate exposure. This can be illustrated by summing the risks associated with exposure to sediments contaminated with PAHs and phthalate compounds. Exposure to 1.6 ppm PAH and 57 ppm total phthalates has a combined risk of 2.6×10^{-6} . The contribution of PAH exposure to the total risk is almost 100 percent (2.6×10^{-6} for PAH vs. 5.5×10^{-9} for phthalate esters). This indicates that an analysis of areas where both PAHs and phthalates are detected at higher concentrations than PCBs is not necessary, since the total risk will be approximately equal to the risk from PAHs alone.

4.0 CONCLUSIONS

Based on Jordan's review of the extent and distribution of organic contamination in sediment, it appears that PAH compounds occur in certain locations and at concentrations which may significantly contribute to the overall environmental and public health risk associated with exposure to sediments at New Bedford Harbor. Based on the evaluation of the extent and level of phthalate ester contamination within the estuary and harbor area, these compounds do not present a significant contribution to either public health or environmental risks. The area identified as presenting an increased risk from PAH contamination is located on the western shore, north of the Coggeshall Street Bridge. This area presents an increased risk to both human health and the environment. Based on this review, a risk estimate developed for the estuary and based solely on the extent and distribution of PCB contamination may underestimate the overall public health and environmental risk resulting from concurrent exposure to PAH compounds. For this reason, Jordan recommends that PAH compounds be considered when assessing the environmental and public health risks associated with exposure to sediments in the estuary.

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